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Eur J Immunol. 1997 Sep;27(9):2330-9.

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☐ 2: Adorini L, Trembleau S.

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Immune deviation towards Th2 inhibits Th-1-mediated autoimmune diabetes:

Biochem Soc Trans. 1997 May;25(2):625-9. Review. No abstract available.

PMID: 9191169 [PubMed - indexed for MEDLINE]

☐ 3: Adorini L, Gregori S, Magram J, Trembleau S.

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The role of IL-12 in the pathogenesis of Th1 cell-mediated autoimmune diseases.

Ann N Y Acad Sci. 1996 Oct 31;795:208-15. Review. No abstract available.

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☐ 4: Trembleau S, Germann T, Gately MK, Adorini L.

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The role of IL-12 in the induction of organ-specific autoimmune diseases.

Immunol Today. 1995 Aug;16(8):383-6. Review.

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☐ 1: Immunol Today. 1995 Aug;16(8):383-6.[Related Articles, Link](#)**ELSEVIER SCIENCE**
FULL-TEXT ARTICLE

The role of IL-12 in the induction of organ-specific autoimmune diseases.

Trembleau S, Germann T, Gately MK, Adorini L.

Roche Milano Ricerche, Italy.

The concept that T cells are subdivided into T helper 1 (Th1) and Th2 subset was recently extended to suggest that Th1 cells contribute to the pathogenesis of several organ-specific autoimmune diseases, whereas Th2 cells inhibit disease development. Here, Sylvie Trembleau and colleagues examine the role of interleukin 12 (IL-12), a key cytokine guiding the development of Th cells, in the induction of autoimmune diseases, and discuss potential immunointervention strategies based on administration of IL-12 antagonists.

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- Review
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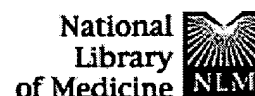


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Deviation of pancreas-infiltrating cells to Th2 by interleukin-12 antagonist administration inhibits autoimmune diabetes.

Trembleau S, Penna G, Gregori S, Gately MK, Adorini L.

Roche Milano Ricerche, Milano, Italy.

Nonobese diabetic (NOD) mice develop spontaneous insulin-dependent diabetes mellitus (IDDM), and the pancreas-infiltrating T cells invariably show a Th1 phenotype. We demonstrated here that the interleukin (IL)-12 antagonist (p40)2 can deviate the default Th1 development of naive T cell receptor (TCR)-transgenic CD4+ cells to the Th2 pathway in vitro. Although (p40)2 does not modify the cytokine profile of polarized Th1 cells, it prevent further recruitment of CD4- cells into the Th1 subset. To study the involvement of Th1 and Th2 cells in the initiation and progression of IDDM, we targeted endogenous IL-12 by administration of (p40)2 in NOD mice. (p40)2 administration to NOD mice inhibits interferon-gamma but not IL-10 production in response to lipopolysaccharide (LPS) or to the putative autoantigen IA-2. Serum immunoglobulin isotypes determined after (p40)2 treatment indicate an increase in Th2 and a decrease in Th1 helper activity. Administration of (p40)2 from 3 weeks of age onwards, before the onset of insulinitis, results in the deviation of pancreas-infiltrating CD4+ but not CD8+ cells to the Th2 phenotype as well as in the reduction of spontaneous and cyclophosphamide-accelerated IDDM. After treating NOD mice with (p40)2 from 9 weeks of age, when insulinitis is well established, few Th2 and a reduced percentage of Th1 cells are found in the pancreas. This is associated with a slightly decreased incidence of spontaneous IDDM, but no protection from cyclophosphamide-accelerated IDDM. In conclusion, deviation of pancreas-infiltrating CD4+ cells to Th2 is associated with protection from IDDM. However, targeting IL-12 after the onset of insulinitis, when the pancreas contains polarized Th1 cells, is not sufficient to induce an effective immune deviation able to significantly modify the course of disease.

PMID: 9341777 [PubMed - indexed for MEDLINE]

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